

CHAPTER 51

Palpation and Percussion of the Abdomen

KEY TEACHING POINTS

- Palpability of the liver depends more on its consistency than size. Firmer livers are easier to palpate. A firm liver increases probability of cirrhosis.
- A palpable spleen is diagnostically helpful in several settings: it increases the probability of malaria in febrile returning travelers, of hepatocellular disease in patients with jaundice, and of hematologic disease in patients with prolonged unexplained fevers.
- In patients with jaundice, a palpable gallbladder increases probability of extrahepatic obstruction (Courvoisier sign), from either malignant or benign causes.
- In patients with increased abdominal girth, two findings *increase* probability of ascites: a positive fluid wave and presence of edema. Two findings *decrease* probability of ascites: flank tympany and absence of edema.
- An expansile pulsating epigastric mass increases probability of an abdominal aortic aneurysm. Nonetheless, many patients with aneurysms lack this finding, especially if their abdominal girth is large or the aneurysm is small.

I. INTRODUCTORY COMMENTS ON TECHNIQUE

Palpation of the abdomen may reveal abnormal tenderness, tumors, hernias, aneurysms, or organomegaly (i.e., of the liver, spleen, or gallbladder). To help the patient relax and to minimize pain during palpation, experienced clinicians recommend that the clinician's hands should be warm, the technique soft and gentle, and the expected tender areas palpated last. Other maneuvers designed to help the patient relax include drawing up the patient's knees, encouraging deep breathing, or engaging the patient in conversation.

In the days before clinical imaging, palpation of a relaxed abdomen was so essential that patients with tense abdominal muscles were often reexamined after immersion in a hot bath or after anesthesia had been induced with ether or chloroform, to determine whether an abnormality was present or not.¹

II. LIVER

A. LIVER SPAN

I. THE FINDING

The liver span is the distance in centimeters between the upper border of the liver in the right midclavicular line (as determined by percussion, i.e., where lung

resonance changes to liver dullness) and the lower border (as determined by either percussion or palpation). Clinicians have been measuring the liver span ever since Piorry introduced topographic percussion in 1828,² although after introduction of the x-ray it became apparent that the estimated span often differed from the actual span, leading most clinicians to adopt the view that the percussed liver span was just an index of liver size, not a precise measurement.³

2. CLINICAL SIGNIFICANCE

The clinician's assessment of liver span usually underestimates the actual value. Clinicians place the upper border too low (2 to 5 cm)^{4,5} and lower border too high (more than 2 cm in approximately half of patients),^{4,6} except in patients with chronic obstructive lung disease, in whom the error with the top border is less.⁴ The liver span is the same whether the patient is percussed during quiet respirations or full held expiration.⁷

Nonetheless, most studies of liver percussion make two points: (1) the estimated span does correlate modestly with actual span, as determined by ultrasonography or scintigraphy ($r = 0.6$ to 0.7).^{3,5,6,8} This correlation is much better in patients with diseased livers than with healthy livers.^{5,8} (2) The percussed liver span is very dependent on the clinician's technique, and consequently, one clinician's "normal liver span" is not the same as another's. The heavier the clinician's percussion stroke, the smaller the measured span and the greater the error in underestimating the actual liver size (see also [Chapter 29](#)).^{4,7} This explains why published estimates of the "normal liver span" range from as low as 6 cm to as high as 15 cm^{6,10-12} and why experienced clinicians, each examining the same patient, differ in their estimate of the patient's span, *on average*, by 8 cm.¹³

These comments imply that each clinician could determine his or her own "normal liver span," based on examination of hundreds of healthy persons, and then use this span as a benchmark to indicate whether a patient's span is abnormally large or not. Nonetheless, two studies applying a standardized percussion technique failed to accurately detect hepatomegaly (likelihood ratio [LR] not significant; [EBM Box 51.1](#)).

B. PALPABLE LIVER EDGE

1. THE FINDING

To palpate the liver edge, the clinician begins by gently palpating the patient's right *lower* quadrant. As the patient breathes in and out, the clinician moves the palpating hand upward 1 to 2 cm at a time, at each location searching for a liver edge that moves down during inspiration and strikes the clinician's fingers. After the edge is located, the clinician should note its consistency (a cirrhotic liver is firmer than a healthy one)⁸ and whether the edge has any irregularities or masses.²⁸

Anatomically the normal liver extends on average 5 cm below the right costal margin at the midclavicular line.⁵

2. CLINICAL SIGNIFICANCE

A. DETECTION OF HEPATOMEGALY

If clinicians palpate what they believe is the patient's liver edge extending below the costal margin, they are virtually always correct (LR = 233.7; see [EBM Box 51.1](#)). Nonetheless, the distance between the liver edge and costal margin correlates poorly with overall liver size, and the finding of a palpable liver edge is an unreliable sign of hepatomegaly (LR is only 1.9; see [EBM Box 51.1](#)). Moreover, approximately

*The normal upper limit for the cephalocaudad dimension of the liver on ultrasonography, from its lower border in the midclavicular line to its upper margin with the lung, is 13 cm.⁹

**EBM BOX 51.1***Detection of Enlarged Liver and Spleen**

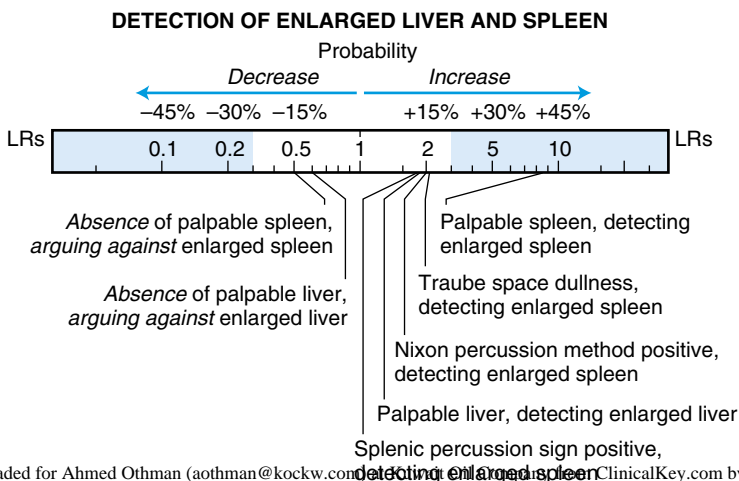
Finding (Reference) [†]	Sensitivity (%)	Specificity (%)	Likelihood Ratio [‡] if Finding Is	
			Present	Absent
Liver				
Percussion Span ≥10 cm in MCL				
Detecting enlarged liver ^{6,14}	61-92	30-43	NS	NS
Palpable Liver				
Detecting liver edge below costal margin ¹⁵	48	100	233.7	0.5
Detecting enlarged liver ^{14,16-18}	39-71	56-85	1.9	0.6
Spleen				
Palpable Spleen				
Detecting enlarged spleen ^{16,17,19-26}	18-78	89-99	8.5	0.5
Splenic Percussion Signs				
Detecting enlarged spleen ^{20,21,25-27}				
Spleen percussion sign	25-85	32-94	1.7	0.7
Nixon method	25-66	68-95	2.0	0.7
Traube space dullness	11-76	63-95	2.1	0.8

*Diagnostic standard: for *enlarged liver*, liver enlarged by scintigraphy,^{16,18} craniocaudal span > 13 cm by ultrasonography,^{6,14} or postmortem weight of liver > 2000 g;¹⁷ for *enlarged spleen*, spleen enlarged by ultrasonography,^{21,24-27} scintigraphy,^{16,19,20,22} or postmortem weight > 200 g¹⁷ or > 250 g.²³

[†]Definition of findings: for *percussed liver span*, using light percussion technique; for *splenic percussion signs*, see the text.

[‡]Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR. MCL, Middlclavicular line; NS, not significant.

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half of livers that extend below the costal margin are not palpable.^{8,15} The consistency of the liver parenchyma probably determines in part whether a liver is palpable, because in patients with cirrhosis, whose livers are smaller but firmer than normal, the liver's edge is palpable 95% of the time.⁸

B. PALPABLE LIVER AND OTHER DISORDERS

In patients with chronic liver disease a few findings modestly increase the probability of cirrhosis: enlarged palpable liver edge (LR = 2.3; EBM Box 51.2), palpable liver in the epigastrium (LR = 2.7), and a liver edge that is unusually firm (LR = 3.3). In patients with jaundice the findings of a palpable liver and liver tenderness are unhelpful, both appearing equally often in patients with hepatocellular disease (i.e., nonobstructive jaundice) as in those with obstructive jaundice (LR not significant; see Chapter 8). In patients with lymphadenopathy the finding of palpable liver fails to distinguish those with serious infections and malignancies from those with benign self-limited disorders (LR not significant; see Chapter 27).

The clinician's assessment of stiffness or firmness of the liver, determined by palpation, correlates very well with noninvasive measures of liver fibrosis, such as ultrasound-based elastography.⁵³



EBM BOX 51.2

Palpation of Liver and Spleen in Various Disorders*

Finding (Reference)	Sensitivity (%)	Specificity (%)	Likelihood Ratio [†] if Finding Is	
			Present	Absent
Liver				
Enlarged palpable liver in patients with chronic liver disease, detecting cirrhosis ²⁹⁻³⁶	31-96	20-96	2.3	0.6
Palpable liver in epigastrium in patients with chronic liver disease, detecting cirrhosis ^{34,36}	50-86	68-88	2.7	0.3
Liver edge firm to palpation in patients with chronic liver disease, detecting cirrhosis ^{30,33,37}	71-78	71-90	3.3	0.4
Palpable liver in patients with jaundice, detecting hepatocellular disease (nonobstructive jaundice) ^{38, 39}	71-83	15-17	NS	NS
Liver tenderness in patients with jaundice, detecting hepatocellular disease (non-obstructive jaundice) ^{38,39}	37-38	70-78	NS	NS
Palpable liver in patients with lymphadenopathy, detecting serious disease ^{40,41}	14-16	86-89	NS	NS

**EBM BOX 51.2***Palpation of Liver and Spleen in Various Disorders—cont'd*

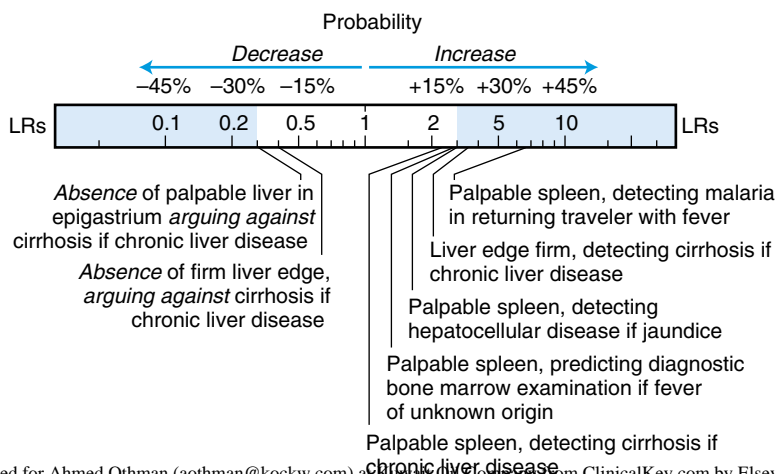
Finding (Reference)	Sensitivity (%)	Specificity (%)	Likelihood Ratio [†] if Finding Is		
			Present	Absent	
Spleen					
Palpable spleen in returning travelers with fever, detecting malaria ⁴²⁻⁴⁴	19-25	95-98	6.5	0.8	
Palpable spleen in patients with jaundice, detecting hepatocellular disease (non-obstructive jaundice) ^{38,39}	29-47	83-90	2.9	0.7	
Palpable spleen in patients with chronic liver disease, detecting cirrhosis ^{30-36,45-48}	5-85	35-100	2.5	0.8	
Palpable spleen in patients with lymphadenopathy, detecting serious disease ^{40,41,49}	5-10	92-96	NS	NS	
Palpable spleen in patients with fever of unknown origin, predicting diagnostic bone marrow examination ⁵⁰⁻⁵²	35-53	82-89	2.9	0.7	

*Diagnostic standard: for *nonobstructive* (vs. *obstructive*) jaundice, needle biopsy of liver, surgical exploration, or autopsy; for *cirrhosis*, needle biopsy of liver (see Chapter 8); for *serious disease* (in patients with lymphadenopathy), see Chapter 27.

[†]Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

MCL, Midclavicular line; NS, not significant.

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PALPATION OF LIVER AND SPLEEN IN VARIOUS DISORDERS

C. AUSCULTATORY PERCUSSION—SCRATCH TEST

I. THE FINDING

Auscultatory percussion (see also [Chapter 29](#)) is frequently used to locate the lower border of the liver. According to traditional teachings, the moment the clinician's percussing digit crosses the border of the liver and begins to strike abdominal wall over the liver, the sound heard through the stethoscope becomes louder.

Nonetheless, the lack of consensus on the proper technique of locating the liver will quickly discourage the serious student of auscultatory percussion. Various experts recommend placing the stethoscope on the xiphoid,^{4,54} near the umbilicus,⁵⁵ superior to⁵⁶ or at the costal margin,⁵⁷ at four separate positions over the liver,⁵⁸ or above the suspected center.⁵⁹ According to various authorities, the clinician should percuss with a finger and pleximeter,⁵⁹ a finger alone,⁵⁶ a bristle brush,⁵⁸ or a corrugated rod.⁵⁸ The direction of the stroke should be circular,¹ centripetal,⁵⁹ centrifugal,⁵⁸ left to right,⁵⁷ or always in a longitudinal axis and toward the liver.^{4,56}

2. CLINICAL SIGNIFICANCE

The evidence supporting auscultatory percussion of the liver is mixed and meager. Only one study supports the technique, showing that 78% of estimates of the lower border are within 2 cm of the actual border (by ultrasonography), compared with 44% for conventional percussion and palpation.⁵⁶ Another study showed that palpation of the liver was more accurate than auscultatory percussion.⁴ A third study showed that there was no correlation whatsoever between the distance of the liver edge below the costal margin, located by auscultatory percussion, and the actual distance (by ultrasonography) for any of 11 different examiners.⁵⁴

D. PULSATILE LIVER

The finding of a pulsatile liver has been described in tricuspid regurgitation with high pulmonary pressures (see [Chapter 46](#)) and constrictive pericarditis.^{60,61} In patients with the holosystolic murmur of tricuspid regurgitation, the finding of a pulsatile liver increases the probability that the regurgitation is moderate to severe (LR = 6.5; see EBM Box 46.1).

III. THE SPLEEN

A. PALPABLE SPLEEN

I. THE FINDING

Experts recommend many different ways to palpate the spleen: some palpate from the patient's right side and others from the patient's left side (curling the fingers over the costal margin to "hook" the spleen edge); some position the patient supine, others position the patient supine with the patient's left fist under his or her left posterior chest, and still others position the patient in the right lateral decubitus position. One study comparing the different positions found all three to be equivalent²¹; the approach clinicians use probably depends most on personal preference.

2. CLINICAL SIGNIFICANCE

A. DETECTION OF SPLENOMEGALY

EBM Box 51.1 indicates that the finding of a palpable spleen increases greatly the probability of splenomegaly (LR = 8.5; see [EBM Box 51.1](#)). Although many enlarged spleens

are not palpable (sensitivity is only 18% to 78%), virtually all massively enlarged spleens (i.e., weight >1 kg or scintigraphic span >22 cm) are detectable by palpation.^{23,62}

B. ETIOLOGY OF SPLENOMEGALY

The common causes of splenomegaly are hepatic disease (i.e., portal hypertension), hematologic disorders (e.g., leukemias, lymphomas, myelofibrosis), infectious disease (e.g., human immunodeficiency virus [HIV] infection), and primary splenic disorders (e.g., splenic infarction or hematoma).^{63,64} The presence of left upper quadrant tenderness and pain increases the probability of a primary splenic disorder or hematologic disorder.⁶⁴ Associated lymphadenopathy practically excludes hepatic disease and points to one of the other disorders (LR = 0.04).⁶⁴ The finding of a palpable liver increases probability of underlying hepatic cause of splenomegaly (LR = 2.7), and the finding of massive splenomegaly (i.e., spleen extends to level of umbilicus) increases the probability of underlying hematologic disease (LR = 2.1).⁶⁴

C. PALPABLE SPLEEN AND OTHER DISORDERS

In returning travelers from tropical countries who are febrile, the finding of a palpable spleen significantly increases the probability of malaria (LR = 6.5; see [EBM Box 51.2](#)). In patients with jaundice the palpable spleen modestly increases probability of hepatocellular disease (i.e., nonobstructive jaundice, LR = 2.9; see [Chapter 8](#)), and in patients with chronic liver disease it increases probability of cirrhosis (LR = 2.5). In patients with lymphadenopathy a palpable spleen is found just as often in patients with serious infections and malignancies as in those with benign, self-limited disorders (LR not significant; see [Chapter 27](#)). In patients with fever of unknown origin (i.e., unexplained fever lasting more than 3 weeks), the finding of a palpable spleen increases probability that a bone marrow biopsy will be diagnostic (LR = 2.9).

B. SPLENIC PERCUSSION SIGNS

I. THE FINDINGS

There are three commonly used splenic percussion signs:

A. SPLEEN PERCUSSION SIGN

Castell described this sign in 1967,¹¹ finding it a useful way to measure splenic size in patients with infectious mononucleosis. The clinician percusses the lowest left intercostal space in the anterior axillary line (usually the eighth or ninth); if the percussion note in this location, usually resonant, becomes dull with a full inspiration, the test is positive. Since Castell's original description, other investigators have regarded any dullness at this location as a positive response (i.e., whether during inspiration or expiration).

B. NIXON METHOD

Nixon described this sign in 1954,⁶⁵ finding it accurate in his experience of 60 splenic aspiration biopsies. The patient is positioned in the right lateral decubitus position, and the clinician percusses from the lower level of pulmonary resonance in the posterior axillary line downwards obliquely to the lower midanterior costal margin. The test is positive if the border of dullness on this line lies more than 8 cm from the costal margin.

C. TRAUBE SPACE DULLNESS

Traube space is the triangular space, normally tympanic, that is over the left lower anterior part of the chest. Its upper border is marked by the limits of cardiac dullness (usually the sixth rib), its lower border is the costal margin, and its lateral border is the anterior axillary line. Although Traube suggested that dullness in this space was a sign of pleural effusion,⁶⁶ Parrino in 1987 suggested that it could be a sign of splenic enlargement.⁶⁷

2. CLINICAL SIGNIFICANCE

Positive percussion signs are much less convincing than palpation (positive LR = 1.7 to 2.1; see [EBM Box 51.1](#)). Traube space dullness becomes even less accurate in overweight patients or those who have recently eaten.⁶⁸

IV. GALLBLADDER: COURVOISIER SIGN

A. THE FINDING

Courvoisier sign is a *palpable nontender* gallbladder in a patient with *jaundice*, a finding that has been traditionally associated with malignant obstruction of the biliary system. Many textbooks call the sign **Courvoisier law**, as if the positive result were pathognomonic of malignancy, although the Swiss surgeon Courvoisier originally presented the finding in 1890 as only an interesting observation.⁶⁹ Writing in a monograph on biliary tract disorders, he stated that, among 187 patients with jaundice and common duct obstruction, a dilated gallbladder was found in only 20% of patients with stones, compared with 92% of patients having other disorders, mostly malignancy.⁷⁰

B. CLINICAL SIGNIFICANCE

Summarizing the information about Courvoisier sign is difficult because various authors define the sign differently. Some apply it to patients without jaundice (clearly not what Courvoisier intended)⁷¹; others define the positive sign as any palpable gallbladder, tender or nontender (some patients with cholecystitis have tender enlarged gallbladders)⁷²⁻⁷⁴; and still others expand the positive sign to include a dilated gallbladder discovered during surgery, clinical imaging, or even autopsy.⁷⁵

Restricting analysis to those studies defining the positive sign as a palpable gallbladder in a jaundice patient, [EBM Box 51.3](#) indicates that Courvoisier sign is pathognomonic for extrahepatic obstruction of the biliary system (i.e., stones or malignancy, LR = 26; i.e., *not* hepatocellular jaundice). However, among patients with biliary obstruction, the sign increases probability only modestly for malignancy and against stones (LR = 2.6). In one series of 86 hospitalized patients with distended gallbladders (as detected by computed tomography or at laparotomy, only 46 (53%) were palpable at the bedside: 83% had a malignant cause of the obstruction and 17% a benign one.⁸⁶

Consequently, if there is a “law” to the Courvoisier sign, it is that the palpable gallbladder in a jaundiced patient indicates extrahepatic obstruction, not that the obstruction is necessarily caused by malignancy.

C. PATHOGENESIS

Courvoisier original hypothesis—that the gallbladder of choledocholithiasis fails to dilate because its walls are fibrotic from chronic cholecystitis—is probably incorrect because experiments with gallbladders of jaundiced patients show that both dilated and nondilated gallbladders have similar wall stiffness.⁸⁷ Instead, patients with dilated gallbladders differ from patients without dilated gallbladders in two important ways: Dilated gallbladders are associated with much higher operative intraductal pressures and longer duration of jaundice.

The relationship between duration of jaundice and dilation of gallbladder explains why Courvoisier's original findings are different from the studies in [EBM Box 51.3](#). When analysis is restricted to just those patients with extrahepatic obstruction, the sensitivity of the dilated gallbladder in malignant obstruction today (25% to 55%) is lower than it was for Courvoisier (i.e., 92%)

**EBM BOX 51.3***Palpation of Gallbladder, Bladder, and Aorta**

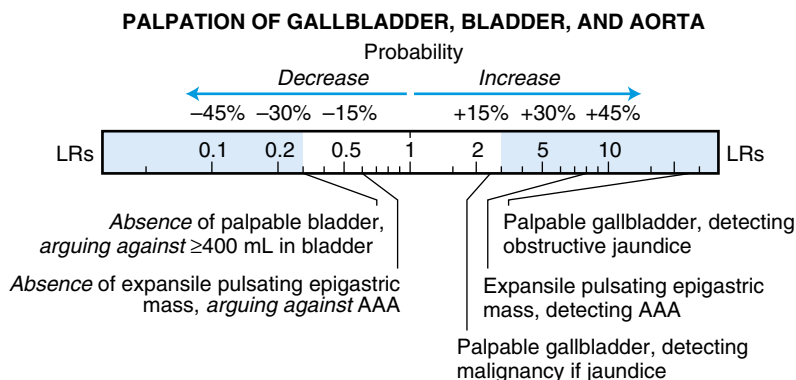
Finding (Reference)	Sensitivity (%)	Specificity (%)	Likelihood Ratio [†] if Finding Is	
			Present	Absent
Gall Bladder				
Palpable Gallbladder				
Detecting obstructed bile ducts in patients with jaundice ³⁸	31	99	26.0	0.7
Detecting malignant obstruction in patients with obstructive jaundice ^{38,71,73,76}	26-55	83-90	2.6	0.7
Bladder				
Palpable Bladder				
Detecting ≥400 mL urine in bladder ⁷⁷	82	56	1.9	0.3
Aorta				
Expansile Pulsating Epigastric Mass				
Detecting abdominal aortic aneurysm (AAA) ⁷⁸⁻⁸⁵	22-68	75-99	8.0	0.6

*Diagnostic standard: for obstructive jaundice and malignant obstruction, needle biopsy of liver, surgical exploration, or autopsy; for ≥400 mL urine in bladder, bladder ultrasound;⁷⁷ for abdominal aortic aneurysm, ultrasonography revealing focal dilation of infrarenal aorta >3 cm in diameter,^{79,80,82-85} >4 cm in diameter,⁸¹ or >1.5 cm larger than proximal aorta.⁷⁸

[†]Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

NS, Not significant.

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(although the specificity is similar at 80% to 90%). The reduced sensitivity may simply reflect that patients with malignant obstruction today, compared with those from a century ago, are diagnosed more quickly using clinical imaging, before pressures increase enough to enlarge the gallbladder greatly.

V. BLADDER VOLUME

For more than a century clinicians have investigated percussing the suprapubic area to detect bladder volume; most studies revealing that the bladder volume must be approximately 400 to 600 mL before dullness reliably appears.⁸⁸ Although the extent of dullness above the symphysis pubis does correlate with bladder volume,^{88,89} overall the sign is unreliable because the results vary tremendously among individual patients and because many patients have inexplicable dullness of the lower abdomen, even without bladder distention.^{2,88}

There are few studies of palpation of the bladder. One study has demonstrated that the *absence* of a palpable bladder in the suprapubic area *decreases* the probability of bladder volumes ≥ 400 mL⁷⁷ (LR = 0.3; see [EBM Box 51.3](#)).

VI. ASCITES

A. THE FINDINGS

In supine patients with ascites, peritoneal fluid gravitates to the flanks and air-filled intestines float to occupy the periumbilical space. This distribution of fluid and air causes four characteristic signs of ascites: (1) Bulging flanks; (2) flank dullness. Flank dullness is positive if there is a *horizontal* border between dullness in the flank area and resonance (or tympany) in the periumbilical area. (3) Shifting dullness. Shifting dullness describes flank dullness that shifts as the patient changes position, usually by rolling on to one side. The sign is based on the principle that air-filled loops of intestine, floating on peritoneal fluid, move to the uppermost position in the abdomen. In a patient with a positive response, the border between resonance and dullness shifts away from the side that is most dependent. To be positive, the shifting border should remain horizontal. (4) Fluid wave. To elicit the fluid wave, the clinician places one hand against the lateral wall of the abdomen and uses the other hand to tap firmly on the opposite lateral wall. In the positive response the tap generates a wave that is transmitted through the abdomen and felt as a sudden shock by the other hand. Because a false-positive response may result from waves travelling through the subcutaneous tissue of the anterior abdominal wall, the clinician should always use the patient's hand or that of an assistant to apply firm pressure against the anterior abdominal wall.

In addition to these four signs, most patients with ascites also have edema, from hypoalbuminemia and the weight of the peritoneal fluid compressing the veins to the legs.⁹⁰

B. PATHOGENESIS

In experiments with cadavers performed a century ago, Müller showed that 1000 mL of fluid injected into the peritoneal space was undetectable by physical examination (i.e., flank or shifting dullness), 1500 mL resulted in some flank dullness, and 2000 mL was the smallest volume to cause shifting dullness.⁸⁸ The living abdominal wall is probably more elastic than the cadaver's, and it is likely that the careful clinician can detect smaller amounts of ascites in patients, but one small study of healthy volunteers still showed that injection of 500 to 1100 mL of fluid was necessary before

shifting dullness appeared.⁹¹ A significant cause of false-positive flank dullness or shifting dullness is accumulation of fluid within loops of the colon.^{91,92} This condition, called *pseudoascites* in the days before clinical imaging,⁹² typically occurred in patients with diarrheal illnesses.

C. CLINICAL SIGNIFICANCE

In patients with abdominal distention the findings *increasing* probability of ascites the most are the positive fluid wave (LR = 5; EBM Box 51.4) and presence of edema (LR = 3.8). The findings *decreasing* probability of ascites the most are *absence* of edema (LR = 0.2) and *absence* of flank dullness (LR = 0.3). Shifting dullness shifts probability of ascites modestly upward when present (LR = 2.3) and modestly downward when absent (LR = 0.4). Findings having relatively little diagnostic value are positive flank dullness, positive bulging flanks, and negative fluid wave. The finding of a flat or everted umbilicus was also diagnostically unhelpful in one study.⁹⁴



EBM BOX 51.4

Ascites*

Finding (Reference) [†]	Sensitivity (%)	Specificity (%)	Likelihood Ratio [‡] if Finding Is	
			Present	Absent
<i>Inspection</i>				
Bulging flanks ⁹³⁻⁹⁵	73-93	44-70	1.9	0.4
Edema ⁹⁴	87	77	3.8	0.2
<i>Palpation and Percussion</i>				
Flank dullness ^{93,94}	80-94	29-69	NS	0.3
Shifting dullness ⁹³⁻⁹⁵	60-87	56-90	2.3	0.4
Fluid wave ⁹³⁻⁹⁵	50-80	82-92	5.0	0.5

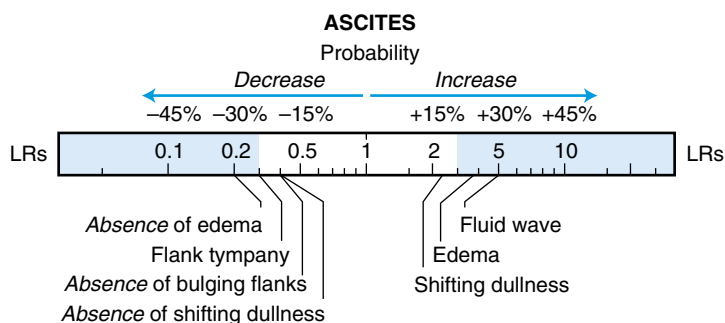
*Diagnostic standard: for ascites, peritoneal fluid by ultrasonography.

[†]Definition of findings: for *shifting dullness*, border between resonance and dullness "shifts" when patient rolls from supine to left lateral decubitus position or right lateral decubitus position; Cattau required a shift in both positions,⁹³ Simel in only one of two positions,⁹⁴ and Cummings used only the right lateral decubitus position at 45 degrees and required a shift > 1 cm.⁹⁵

[‡]Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

NS, Not significant.

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Auscultatory percussion also has been recommended to detect ascites,⁹⁶⁻⁹⁸ although only the puddle sign (auscultatory percussion of the prone patient) has been formally tested,⁹³⁻⁹⁴ proving to be diagnostically unhelpful.

VII. ABDOMINAL AORTIC ANEURYSM

A. INTRODUCTION

Abdominal aortic aneurysm is a focal ballooning of the infrarenal abdominal aorta, traditionally defined as a diameter greater than 3 to 4 cm. It is a disorder of elderly patients, affecting 1% to 2% of patients over the age of 50.^{99,100} Abdominal aortic aneurysms tend to enlarge slowly, but some rupture catastrophically with an overall mortality of up to 90%.¹⁰¹

B. THE FINDING

Because the normal aorta bifurcates at the level of the umbilicus, palpable aortic aneurysms usually are found in the epigastrium or left upper quadrant. The clinician should place one hand on each side of the aorta and measure its diameter, subtracting the estimated thickness of two layers of skin and subcutaneous tissue. Most studies do not specifically define the positive finding (instead stating simply the positive finding is “aortic aneurysm present by palpation”), although others define it as an estimated diameter greater than 3 cm using the previously described method.⁷⁹

Importantly, an aortic aneurysm pushes the two hands *apart*, a finding called *expansile* pulsation.¹⁰² Other prominent epigastric pulsations sometimes occur in patients with thin abdomens or in those with epigastric masses overlying the normal aorta, but unless these pulsations are *expansile*, they do not indicate an aneurysm.

C. CLINICAL SIGNIFICANCE

According to [EBM Box 51.3](#) the finding of a palpable epigastric pulsation suggestive of aneurysm increases probability that one is present (LR = 8; see [EBM Box 51.3](#)). In contrast, the absence of this finding is much less helpful (LR is only 0.6), simply because the sensitivity for the finding is as low as 22% (i.e., up to 78% of patients with aneurysms lack a prominent pulsation).

The two most important variables governing whether an aneurysm is palpable are the size of the aneurysm and the girth of the patient's abdomen. Aneurysms between 3 and 5 cm in diameter are difficult to detect, and if *aneurysm* is instead defined as a focal bulging more than 5 cm in diameter—the diameter usually indicating surgical repair—the sensitivity of bedside examination increases to more than 80% in almost all series.^{79,100,103} Aneurysms are also more difficult to detect in patients with larger abdominal girths.^{78,79,103,104} After restricting the analysis to just patients with abdominal girth of less than 100 cm (measured at the umbilicus)^{78,79} or to patients in whom the clinician can palpate the aorta,^{79,104,105} the sensitivity of the examination exceeds 88% in all studies. These results indicate that the negative examination significantly decreases probability of an aneurysm of more than 5 cm in diameter, especially if the patient has a girth of less than 100 cm or has a palpable aorta.

The most common cause for a false-positive examination is an abnormally tortuous aorta.^{106,107} Rare causes are a horseshoe kidney, intra-abdominal tumor, or paraaortic adenopathy.^{106,107}

The references for this chapter can be found on www.expertconsult.com.

REFERENCES

1. Cabot RC. *Physical Diagnosis*. New York, NY: William Wood and Co.; 1926.
2. McGee S. Percussion and physical diagnosis: separating myth from science. *Disease-a-Month*. 1995;41(10):643–692.
3. Zelman S, Pickard CM. Roentgen and autopsy evaluation of percussion of the liver and spleen. *Gastroenterology*. 1955;29:1037–1045.
4. Sullivan S, Krasner N, Williams R. The clinical estimation of liver size: a comparison of techniques and an analysis of the source of error. *Br Med J*. 1976;2:1042–1043.
5. Peternel WW, Schaefer JW, Schiff L. Clinical evaluation of liver size and hepatic scintiscan. *Am J Dig Dis*. 1966;11(5):346–350.
6. Sapira JD, Williamson DL. How big is the normal liver? *Arch Intern Med*. 1979;139:971–973.
7. Castell DO, O'Brien KD, Muench H, Chalmers TC. Estimation of liver size by percussion in normal individuals. *Ann Intern Med*. 1969;70:1183–1189.
8. Zoli M, Magalotti D, Grimaldi M, Gueli C, Marchesini G, Pisi E. Physical examination of the liver: is it still worth it? *Am J Gastroenterol*. 1995;90(9):1428–1432.
9. Niederau C, Sonnenberg A, Mueller JE, Erckenbrecht JF, Scholten T, Fritsch WP. Sonographic measurements of the normal liver, spleen, pancreas, and portal vein. *Radiology*. 1983;149:537–540.
10. Goodman JL. The enlarged liver in diabetes mellitus: its determination by percussion. *Am J Digest Dis*. 1950;18:181–185.
11. Castell DO. The spleen percussion sign: a useful diagnostic technique. *Ann Intern Med*. 1967;67(6):1265–1267.
12. Naftalis J, Leevy CM. Clinical estimation of liver size. *Am J Dig Dis*. 1963;8(3):236–243.
13. Blendis LM, McNeilly WJ, Sheppard L, Williams R, Laws JW. Observer variation in the clinical and radiological assessment of hepatosplenomegaly. *Br Med J*. 1970;1:727–730.
14. Joshi R, Singh A, Jajoo N, Pai M, Kalantri SP. Accuracy and reliability of palpation and percussion for detecting hepatomegaly: a rural hospital-based study. *Indian J Gastroenterol*. 2004;23:171–173.
15. Ariel IM, Briceno M. The disparity of the size of the liver as determined by physical examination and by hepatic gammascanning in 504 patients. *Med Pediatr Oncol*. 1976;2:69–73.
16. Halpern S, Coel M, Ashburn W, et al. Correlation of liver and spleen size: determinations by nuclear medicine studies and physical examination. *Arch Intern Med*. 1974;134:123–124.
17. Riemenschneider PA, Whalen JP. The relative accuracy of estimation of enlargement of the liver and spleen by radiologic and clinical methods. *Am J Roentgenol Radium Ther Nucl Med*. 1965;94:462–468.
18. Rosenfield AT, Laufer I, Schneider PB. The significance of a palpable liver: a correlation of clinical and radioisotope studies. *Am J Roentgenol Radium Ther Nucl Med*. 1974;122:313–317.
19. Westin J, Lanner L, Larsson A, Weinfeld A. Spleen size in polycythemia: a clinical and scintigraphic study. *Acta Med Scand*. 1972;191:263–271.
20. Sullivan S, Williams R. Reliability of clinical techniques for detecting splenic enlargement. *Br Med J*. 1976;2:1043–1044.
21. Barkun AN, Camus M, Green L, et al. The bedside assessment of splenic enlargement. *Am J Med*. 1991;91:512–518.
22. Holzbach RT, Clark RE, Shipley RA, Kent 3rd WB, Lindsay GE. Evaluation of spleen size by radioactive scanning. *J Lab Clin Med*. 1962;60(6):902–913.
23. Ingeberg S, Stockel M, Sorensen PJ. Prediction of spleen size by routine radioisotope scintigraphy. *Acta Haemat*. 1983;69:243–248.
24. Gerspacher-Lara R, Pinto-Silva RA, Serufo JC, Rayes AAM, Drummond SC, Lambertucci JR. Splenic palpation for the evaluation of morbidity due to Schistosomiasis mansoni. *Mem Inst Oswaldo Cruz*. 1998;93(suppl 1):245–248.
25. Dubey S, Swaroop A, Jain R, Verma K, Garg P, Agarwal S. Percussion of Traube's space. A useful index of splenic enlargement. *J Assoc Phys India*. 2000;48:326–328.
26. Chongtham DS, Singh MM, Kalantri SP, Pathak S. Accuracy of palpation and percussion manoeuvres in the diagnosis of splenomegaly. *Indian J Med Sci*. 1997;51(11):409–416.

27. Tamayo SG, Rickman LS, Mathews WC, et al. Examiner dependence on physical diagnostic tests for the detection of splenomegaly: a prospective study with multiple observers. *J Gen Intern Med.* 1993;8:69–75.
28. Fenster F, Klatskin G. Manifestations of metastatic tumors of the liver: a study of eighty-one patients subjected to needle biopsy. *Am J Med.* 1961;31:238–248.
29. Hamberg KJ, Carstensen B, Sorensen TIA, Eghoe K. Accuracy of clinical diagnosis of cirrhosis among alcohol-abusing men. *J Clin Epidemiol.* 1996;49(11):1295–1301.
30. Marmo R, Romano M, Peduto A, Caporaso N, Persico M, Coltorti M. Decision-making model for a non-invasive diagnosis of compensated liver cirrhosis. *Ital J Gastroenterol.* 1993;25:1–8.
31. Nakamura T, Nakamura S, Aikawa T, Suzuki O, Onodera A, Karoji N. Clinical studies of alcoholic hepatic diseases. *Tohoku J Exp Med.* 1967;93:179–189.
32. Rankin JGD, Orrego-Matte H, Deschenes J, Medline A, Findlay JE, Armstrong AIM. Alcoholic liver disease: the problem of diagnosis. *Alcohol Clin Exp Res.* 1978;2(4):327–338.
33. Tine F, Caltagirone M, Camma C, et al. Clinical indicants of compensated cirrhosis: a prospective study. In: Dianzani MU, Gentilini P, eds. *Chronic Liver Damage: proceedings of the Annual Meeting of the Italian National Programme on Liver Cirrhosis.* Amsterdam: Excerpta Medica; 1990:187–198. San Miniato, Italy 11–13 January 1990.
34. McCormick PA, Nolan N. Palpable epigastric liver as a physical sign of cirrhosis: a prospective study. *Eur J Gastroenterol Hepatol.* 2004;16:1331–1334.
35. Gordon A, Bailey MJ, Gibson PR, Roberts SK. Comprehensive clinical assessment improves the accuracy of predicting cirrhosis in chronic hepatitis C. *J Gastroenterol Hepatol.* 2005;20:825–832.
36. Romagnuolo J, Jhangri GS, Jewell LD, Bain VG. Predicting the liver histology in chronic hepatitis C: how good is the clinician? *Am J Gastroenterol.* 2001;96(11):3165–3174.
37. Aubé C, Winkfield B, Oberti F, et al. New doppler ultrasound signs improve the non-invasive diagnosis of cirrhosis or severe liver fibrosis. *Eur J Gastroenterol Hepatol.* 2004;16:743–751.
38. Schenker S, Balint J, Schiff L. Differential diagnosis of jaundice: report of a prospective study of 61 proved cases. *Am J Dig Dis.* 1962;7(5):449–463.
39. Burbank F. A computer diagnostic system for the diagnosis of prolonged undifferentiating liver disease. *Am J Med.* 1969;46:401–415.
40. Vassilakopoulos TP, Pangalis GA. Application of a prediction rule to select which patients presenting with lymphadenopathy should undergo a lymph node biopsy. *Medicine.* 2000;79:338–347.
41. Slap GB, Brooks JSJ, Schwartz JS. When to perform biopsies of enlarged peripheral lymph nodes in young patients. *J Am Med Assoc.* 1984;252(10):1321–1326.
42. O'Brien D, Tobin S, Brown GV, Torresi J. Fever in returned travelers: review of hospital admissions for a 3-year period. *Clin Infect Dis.* 2001;33:603–609.
43. Bottieau E, Clerinx J, Van den Enden E, et al. Fever after a stay in the tropics: diagnostic predictors of the leading tropical conditions. *Medicine.* 2007;86(1):18–25.
44. D'Acromont V, Landry P, Mueller I, Pecoud A, Genton B. Clinical and laboratory predictors of imported malaria in an outpatient setting: an aid to medical decision making in returning travelers with fever. *Am J Trop Med Hyg.* 2002;66(5):481–486.
45. Cozzolino G, Lonardo A, Francica G, Amendola F, Cacciatore L. Differential diagnosis between hepatic cirrhosis and chronic active hepatitis: specificity and sensitivity of physical and laboratory findings in a series from the Mediterranean area. *Am J Gastroenterol.* 1983;78(7):442–445.
46. Czaja AJ, Wolf AM, Baggenstoss AH. Clinical assessment of cirrhosis in severe chronic active liver disease: specificity and sensitivity of physical and laboratory findings. *Mayo Clin Proc.* 1980;55:360–364.
47. Hay CRM, Preston FE, Trigger DR, Greaves M, Underwood JCE, Westlake L. Predictive markers of chronic liver disease in hemophilia. *Blood.* 1987;69(6):1595–1599.
48. Lashner BA, Jonas RB, Tang HS, Evans AA, Ozeran SE, Baker AL. Chronic hepatitis: disease factors at diagnosis predictive of mortality. *Am J Med.* 1988;85:609–614.
49. Tokuda Y, Kishaba Y, Kato J, Nakazato N. Assessing the validity of a model to identify patients for lymph node biopsy. *Medicine.* 2003;82:414–418.

50. Wang HY, Yag CF, Chiou TJ, et al. A "bone marrow score" for predicting hematological disease in immunocompetent patients with fevers of unknown origin. *Medicine*. 2014;93:e243.
51. Ben-Baruch S, Canaani J, Braunstein R, et al. Predictive parameters for a diagnostic bone marrow biopsy specimen in the work-up of fever of unknown origin. *Mayo Clin Proc*. 2012;87:136–142.
52. Hot A, Jaisson I, Girard C, et al. Yield of bone marrow examination in diagnosing the source of fever of unknown origin. *Arch Intern Med*. 2009;169:2018–2023.
53. Lenci I, Cucchiarelli S, Milana M, Riccobelli F, Baiocchi L. Physical examination of the liver: does it make sense in the third millennium? *Liver Int*. 2013;33:806–807.
54. Tucker WN, Saab S, Rickman LS, Mathews WC. The scratch test is unreliable for detecting the liver edge. *J Clin Gastroenterol*. 1997;25(2):410–414.
55. Kukowka A. Auskultatorische Methode zur bestimmung der Lebergroesse—ein einfaches, probates Schnellverfahren. *Z Allgemeinmedizin*. 1972;48:1645–1646.
56. Fuller GN, Hargreaves MR, King DM. Scratch test in clinical examination of liver. *Lancet*. 1988;1:181.
57. Rinzier SH. Re-emphasis of the auscultatory method for ascertaining the size of the liver. *N Y State J Med*. 1950;50:300.
58. Sehrwald. Ueber die Brauchbarkeit des Phonoendoskopes. *Dtsch Arch Klin Med*. 1904;79:450–467.
59. Camman GP, Clark A. A new mode of ascertaining the dimensions, form, and condition of internal organs by percussion. *N Y J Med Surg*. 1840;3:62–96.
60. El-Sherif A, El-Said G. Jugular, hepatic, and praecordial pulsations in constrictive pericarditis. *Br Heart J*. 1971;33:305–312.
61. Coralli RJ, Crawley IS. Hepatic pulsations in constrictive pericarditis. *Am J Cardiol*. 1986;58:370–373.
62. Arkles LB, Gill GD, Molan MP. A palpable spleen is not necessarily enlarged or pathological. *Med J Austral*. 1986;145:15–17.
63. Lipp WF, Eckstein EH, Aaron AH. The clinical significance of the palpable spleen. *Gastroenterology*. 1944;3:287–291.
64. O'Reilly RA. Splenomegaly at a United States County hospital: diagnostic evaluation of 170 patients. *Am J Med Sci*. 1996;312(4):160–165.
65. Nixon RK. The detection of splenomegaly by percussion. *N Engl J Med*. 1954;250(4):166–167.
66. Verghese A, Krish G, Karnad A. Ludwig Traube: the man and his space. *Arch Intern Med*. 1992;152:701–703.
67. Parrino TA. The art and science of percussion. *Hosp Pract*. 1987;99:25–36.
68. Barkun AN, Camus M, Meagher T, et al. Splenic enlargement and Traube's space: how useful is percussion? *Am J Med*. 1989;87:562–566.
69. Anonymous. Ludwig Courvoisier (1843-1918): courvoisier's sign. *J Am Med Assoc*. 1968;204(7):165.
70. Verghese A, Dison C, Berk SL. Courvoisier's "Law"—a eponym in evolution. *Am J Gastroenterol*. 1987;82(3):248–250.
71. Viteri AL. Courvoisier's law and evaluation of the jaundiced patient. *Tex Med*. 1980;76:60–61.
72. Gunn A, Keddie N. Some clinical observations on patients with gallstones. *Lancet*. 1972;2:239–241.
73. Chen JJ, Changchien CS, Tai DI, Kuo CH. Gallbladder volume in patients with common hepatic duct dilatation: an evaluation of Courvoisier's sign using ultrasonography. *Scand J Gastroenterol*. 1994;29(3):284–288.
74. Fournier AM, Michel J. Courvoisier's sign revisited: two patients with palpable gallbladder. *South Med J*. 1992;85(5):548–550.
75. Mikal S, Campbell AJA. Carcinoma of the pancreas: diagnostic and operative criteria based on one hundred consecutive autopsies. *Surgery*. 1950;28(6):963–969.
76. Zollinger R, Kevorkian AY. Surgical aspects of obstructive jaundice. *N Engl J Med*. 1939;221:486–488.
77. Weatherall M, Harwood M. The accuracy of clinical assessment of bladder volume. *Arch Phys Med Rehabil*. 2002;83:1300–1302.

78. Lederle FA, Walker JM, Reinke DB. Selective screening for abdominal aortic aneurysms with physical examination and ultrasound. *Arch Intern Med.* 1988;148:1753–1756.
79. Fink HA, Lederle FA, Roth CS, Bowles CA, Nelson DB, Haas MA. The accuracy of physical examination to detect abdominal aortic aneurysm. *Arch Intern Med.* 2000;160:833–836.
80. Twomey A, Twomey E, Wilkins RA, Lewis JD. Unrecognised aneurysmal disease in male hypertensive patients. *Inter Angiol.* 1986;5:269–273.
81. Collin J, Walton J, Araujo L, Lindsell D. Oxford screening programme for abdominal aortic aneurysm in men aged 65 to 74 years. *Lancet.* 1988;2:613–615.
82. Allen PIM, Gourevitch D, McKinley J, Tudway D, Goldman M. Population screening for aortic aneurysms. *Lancet.* 1987;2:736.
83. Al Zahrani HA, Rawas M, Maimani A, Gasab M, Al Khail BA. Screening for abdominal aortic aneurysm in the Jeddah area, western Saudi Arabia. *Cardiovasc Surg.* 1996;4(1):87–92.
84. Andersson AP, Ellitsgaard N, Jorgensen B, et al. Screening for abdominal aortic aneurysm in 295 outpatients with intermittent claudication. *Vasc Surg.* 1991;25:516–520.
85. MacSweeney STR, O'Meara M, Alexander C, O'Malley MK, Greenhalgh RM. High prevalence of unsuspected abdominal aortic aneurysm in patients with confirmed symptomatic peripheral or cerebral arterial disease. *Br J Surg.* 1993;80:582–584.
86. Munzer D. Assessment of Courvoisier's law. *Saudi J Gastroenterol.* 1999;5:106–112.
87. Chung RS. Pathogenesis of the "Courvoisier gallbladder". *Dig Dis Sci.* 1983;28(1):33–38.
88. Müller F. Einige Beobachtungen aus dem Percussionskurs. *Berl Klin Wochenschr.* 1895;32:278–280.
89. Guarino JR. Auscultatory percussion of the urinary bladder. *Arch Intern Med.* 1985;145:1823–1825.
90. Hussey HH, Jeghers H. Practical considerations of venous pressure. *N Engl J Med.* 1947;237:776–782. 812–818.
91. Moses WR. Shifting dullness in the abdomen. *South Med J.* 1946;39(12):985–987.
92. Sahli H. *A Treatise on Diagnostic Methods of Examination.* Philadelphia, OA: W. B. Saunders; 1911.
93. Cattau EL, Benjamin SB, Knuff TE, Castell DO. The accuracy of the physical examination in the diagnosis of suspected ascites. *J Am Med Assoc.* 1982;247:1164–1166.
94. Simel DL, Halvorsen RA, Feussner JR. Quantitating bedside diagnosis: clinical evaluation of ascites. *J Gen Intern Med.* 1988;3:423–428.
95. Cummings S, Papadakis M, Melnick J, Gooding GAW, Tierney LM. The predictive value of physical examination for ascites. *West J Med.* 1985;142:633–636.
96. Guarino JR. Auscultatory percussion to detect ascites. *N Engl J Med.* 1986;315:1555–1556.
97. McLean ACJ. Diagnosis of ascites by auscultatory percussion and hand-held ultrasound unit. *Lancet.* 1987;2:1526–1527.
98. Lawson JD, Weissbein AS. The puddle sign—an aid in the diagnosis of minimal ascites. *N Engl J Med.* 1959;260(13):652–654.
99. Lederle FA, Johnson GR, Wilson SE, et al. Prevalence and associations of abdominal aortic aneurysm detected through screening. *Ann Intern Med.* 1997;126:441–449.
100. Lederle FA, Simel DL. Does this patient have abdominal aortic aneurysm? *J Am Med Assoc.* 1999;281:77–82.
101. Ernst CB. Abdominal aortic aneurysm. *N Engl J Med.* 1993;328(16):1167–1172.
102. Osler W. Aneurysm of the abdominal aorta. *Lancet.* 1905;2:1089–1096.
103. Chervu A, Clagett P, Valentine J, Myers SI, Rossi PJ. Role of physical examination in detection of abdominal aortic aneurysms. *Surgery.* 1995;117:454–457.
104. Arnell TD, de Virgilio C, Donayre C, Grant E, Baker JD, White R. Abdominal aortic aneurysm screening in elderly males with atherosclerosis: the value of the physical exam. *Am Surg.* 1996;62:861–864.
105. Cabellon S, Moncrief CL, Pierre DR, Cavanaugh DG. Incidence of abdominal aortic aneurysms in patients with atheromatous arterial disease. *Am J Surg.* 1983;146:575–576.
106. Nusbaum JW, Freimanis AK, Thomford NR. Echography in the diagnosis of abdominal aortic aneurysm. *Arch Surg.* 1971;102:385–388.
107. Robicsek F, Daugherty HK, Mullen DC, Tam W, Scott WP. The value of angiography in the diagnosis of unruptured aneurysms of the abdominal aorta. *Ann Thorac Surg.* 1971;11(6):538–550.